Kindly amend the application as follows:*

IN THE SPECIFICATION

Replace the paragraph on page 24, lines 6-21 with the following:

After the surgical removal of spleens from naive C57BL/6 mice, the spleens were crushed through a sterile grid directly into a dish containing DMEM medium supplemented with 2% FCS. The cells were vigorously suspended, transferred into Falcon tubes, and centrifuged for a few seconds after which the tubes were let stand for 10 minutes. Single spleen cells remained in the supernatant, which was poured off into another tube. The leukocytes were counted by trypan-blue exclusion and adjusted to a cell density of 7x106 viable cells per ml. All three peptides used for pulsing (peptide 1 KTWGQYWQV (SEQ ID NO:5), peptide 2 ITDQVPFSV (SEQ ID NO:6) and peptide 3 VLYRYGSFSV (SEQ ID NO:7)) were purchased from MWG Biotech (Ebersberg, Germany). A 100-fold peptide stock solution in PBS was prepared using several sonification steps to promote dissolution. The peptide solution (10 nM) was added to the spleen cell suspension and incubated for 1 hour at room temperature and for another 1 hour at 37°C under 5%CO2. The peptide-pulsed spleen cells were injected intraperitoneally into the mice (2x107 cells per mouse).

^{*} Applicants attach an Appendix showing the changes that have been made. Underlines indicate an addition. Brackets indicate a deletion.

Replace the paragraph on page 26, lines 1-22 with the following:

Five different immunogenic peptides from the hgp100 protein have been identified previously and shown to be reactive with different hgp100 TILs (Kawakami et al., J. Immunother. 21 (1998), 237-246). Two of the nonamer peptides which were also used in the present application, peptide 1 G9(154-162) and peptide 2 G9(209-217), appeared to be immunodominant and were widely recognized by TILs (Pass et al., Cancer J. Sci. Am. 4 (1998), 316-323; Rivoltini et al., J. Immunol. 156 (1996), 3882-3891; Salgaller et al., Cancer Res. 56 (1996), 4749-4757; Clay et al., J. Immunol. 162 (1999), 1749-1755). Peptide 3, the decamer G10(476-485), is expressed on the surface of melanoma cells and is able to induce melanoma-reactive CTLs from peripheral blood lymphocytes (PBL) of melanoma patients by repeated in vitro stimulation (Salgaller et al., Cancer Res. 55 (1995), 4972-4979). Thus, these gp100 epitopes were considered as good candidates for use in peptide-based immunotherapies. Recently, treatment of melanoma patients with synthetic peptides representing putative CTL-specific epitopes summarized in a review of Rosenberg ((1997), loc. cit.) have shown some success when used for ex vivo pulsing of autologous APCs, for instance dendritic cells which are specialized for the induction of primary T cell response (Nestle et al., Nat. Med. 4 (1998), 328-332). Three of previously described CTL epitopes used for pulsing autologous spleen cells from C57BL/6 mice are indicated in the sequence alignment of human and mouse gp100 (Fig. 1B), peptide 1 (KTWGQYWQV; SEQ ID NO:5), peptide 2 (ITDQVPFSV; SEQ ID NO:6) and peptide 3 (VLYRYGSFSV; SEQ ID NO:7). Peptide 2 is identical between human and mouse, while the peptides 1 and 3 differ in one amino acid.